Characteristics of Functional Auditory Hallucinations

TO THE EDITOR: In schizophrenia, functional hallucinations are defined as those that occur when a patient simultaneously receives a real stimulus in the perceptual field concerned (e.g., hallucinated voices heard simultaneously with—and specific to—the real sound of running water) (1).

Mr. A, a 35-year-old man who had suffered from schizophrenia since his early 20s, was referred for assessment of refractory auditory verbal hallucinations. He was taking quetiapine, having previously been given several other antipsychotics, including clozapine (which was discontinued after a generalized tonic-clonic seizure and neutropenia). No pharmacological treatments had resulted in significant attenuation of his auditory hallucinations (the predominant clinical symptom of his illness).

The most salient hallucinated voice was perceived solely when Mr. A simultaneously heard real engine sounds from motor vehicles. The engine sounds and the voice were perceived as "in parallel." The "engine voice" spoke to him in the second person, uttering frightening statements such as, "I've got hell for you." The timbre of this voice was mechanistic, like the accompanying engine sounds, and lacked human characteristics, such as gender or accent.

Another hallucinated voice occurred simultaneously with actual speech uttered by television announcers. The semantic content was the same as that of the "engine voice," but the "television voice" sounded human, exactly like the real voice of the television announcer who was speaking at the same time. For example, the "television voice" was often described as sounding like an adult woman with a northern British accent and "serious" emotional prosody.

A third variant of Mr. A's functional auditory hallucinations occurred when he played his electric guitar. Single hallucinated words accompanied the playing (and perception) of each individual musical note. These words were in a seemingly random order, without apparent semantic features. Like the "engine voice," the "guitar voice" lacked human attributes but instead sounded like an electric guitar. For example, if Mr. A played a musical scale, then he would hear hallucinated words whose pitch matched the simultaneously perceived guitar notes. A physical examination, routine blood investigations, and an audiological assessment revealed no abnormalities.

In this patient, we observed a direct relationship between the timbre, prosody, and pitch of real environmental sounds and simultaneously perceived auditory hallucinations. Evidence from functional neuroimaging supports a general hypothesis that auditory hallucinations can arise because of abnormal activation in the auditory cortex (2, 3). This case suggests a further hypothesis: normal activation in the auditory system, which corresponds to neural encoding of natural-sound object and location characteristics (4, 5), may be misinterpreted, leading to the false perception of functional auditory hallucinations that *retain* certain acoustic features that were present in the original signal.

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Hepatotoxicity Related to Citalopram

TO THE EDITOR: Selective serotonin reuptake inhibitors (SSRIs) are currently the mainstay therapy for depression because of their favorable side effect profile and safety in overdose. Only isolated cases of liver injury in association with fluoxetine, paroxetine, sertraline, and fluvoxamine have previously been reported (1). To our knowledge, the following case is the first reported instance of significant liver damage related to the SSRI citalopram.

Mr. A, a 44-year-old man, was given a prescription for 100 mg/day of hydroxycine hydrochloride, 2 mg/day of clonazepam, and 20 mg/day of citalopram for depression. Two years earlier, he did well during 6 months of fluoxetine treatment; the results of prior liver function tests were normal. After 8 weeks of treatment, he was seen by us because of 7 days of asthenia and weight loss. He reported no use of alcohol, any other drugs, or herbal remedies and had received no blood transfusions.

The results of a physical examination were normal. His level of aspartate aminotransferase was 277 IU/liter (normal <30), and his alanine aminotransferase level was 1078 IU/liter (normal <36). His bilirubin and alkaline phosphatase levels were normal, as was his eosinophil count. Serology tests ruled out viral causes. Screening for autoantibodies produced negative results, and the results of an abdominal ultrasonographic examination were normal. Treatment with citalopram was stopped while other treatments were maintained intermittently. Five days after drug withdrawal, Mr. A's alanine aminotransferase level fell more than 50%, and a complete return to normal was seen within 2 months.

A causal association between citalopram and hepatocellular injury can confidently be established because there was a temporal relationship between the administration of the drug and the onset of hepatic abnormalities, there was a rapid recovery after stopping the drug, and alternative explanations were ruled out. Of interest, citalopram hepatotoxicity was associated in this patient with nonspecific symptoms. In patients who do not develop jaundice, hepatotoxicity may have an unspecific clinical presentation. This could lead clinicians to attribute these clinical manifestations to worsening de-